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STUDIES ON THE SECOND PHASE OF NYSTAGMUS

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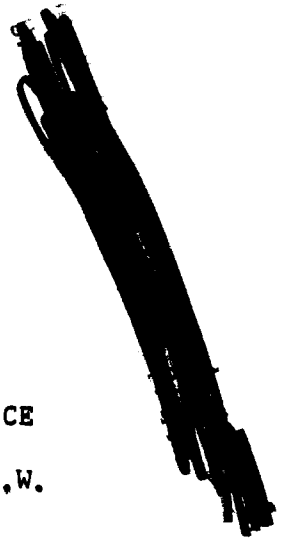

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STUDIES ON THE SECOND PHASE OF NYSTAGMUS

by Akio Noto

(Department of Oto-Rhino-Laryngology, Niigata University School of Medicine)

Nippon Jibiinkoka Gakkai Kaiho (Journal of Oto-Rhino-Laryngol. Soc. Jap.)

PART I: THE SECOND PHASE OF POST-ROTARY NYSTAGMUS

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Introduction

In 1907, Bárány [4] observed that the nystagmus occurring after rotatory stimulation to the vestibular organ should a slow rhythmic movement toward the direction of rotation under certain conditions. He called this nystagmus which occurs after a period of pause following disappearance of the post-rotatory nystagmus "nach-nachnystagmus." Subsequently Buys (1924, 1925) and Fisher (1928) reported the same phenomenon, which have led to intensive studies of the etiologic mechanism of this secondary phase of post-rotatory nystagmus ever since. There have been two opposite hypotheses on the origin of this nystagmus, i. e., central process (Fisher, Dodge, Schmalz, Veits, Mittermaier, Aschan, etc.) and peripheral process (Groen, Jongkees, etc.), but no definite answer has yet been found. The present study was initially aimed at investigating the causal mechanism of the secondary phase of post-rotatory nystagmus. With the progress of the study, however, it became clear that the secondary phase could be observed not only in post-rotatory nystagmus but also in caloric and optokinetic, nystagmus. It has been known, therefore, that the mechanism of this secondary nystagmus should be studied as a phonomenon common to all nystagmus regardless of the kind of stimulus.

The recent progress in the study of vestibular nystagmus is largely attributable to an improvement of the recording method and stimulation apparatus. Since Bugs and Fisher devised an electric rotatory chair as a source of rotatory stimuli, it has become possible to give a constant stimulation to the vestibular organ. As to the recording method, the invention and development of electronystagmography (E.N.G.) has contributed a great deal to the accuracy of recording ocular movement. Schott (1922) first recorded nystagmus by a string galvanometer applying the polarity of electrodes inserted into the palpebral conjunctiva. Subsequently, Meyer (1929) succeeded in the recording by placing electrodes on the external canthi of the eyes. Since then, the method of recording ocular movement by registering changes in the corneo-retinal potential has been improved successively. New findings obtained in the present study are the result of making the most use of the electric rotatory chair and ENG machine having differential circuits.

A. Methods of Experiment

The recording apparatus of nystagmus used in this study is a two-channel E.N.G. machine provided with a differentiator (Sanei Measuring Instrument Co.) the outline of which is shown in Figure 1. Details of this apparatus have been reported by Koike et al. (1958) [18]. It is a direct (ink)-writing oscillograph using a galvanometer.

Particular attention was paid to the calibration of the amplifier so that artifacts due to the polarization potential of electrodes and other factors could easily be recognized. Although a longer time constant is generally desirable for recording nystagmus, it tends to make the recognition of artifacts difficult. In the present study it was determined to use the time constant of 0.6 and 1.5 seconds for recording original waveforms of nystagmus and that of 0.03 and 0.3 second for recording differential waveforms. In human experiments, round silver palte (8 mm in diameter) were used for electrodes. They were placed about 1 cm apart from the external canthi of the eyes. A ground electrode was placed on the forehead. The surface of the skin was cleaned with benzine and the electrodes were closely adhered to the skin with Henriksson's paste and covered with bandages for securing fixation. About 10 minutes after the application of electrodes, the resistance of electrodes were measured. When it was greater than 100 kilo-ohms, the electrode was re-applied. In rabbits, needle electrode of electromyography were used. They were inserted into the subcutaneous tissue about 5 mm apart from the external canthi of the eyes. A ground electrode was placed on the top of the head. Considering the greater susceptibility of these needle electrodes to artifacts, particular attention was paid to the direction and depth of insertion for eliminating artifacts.

After applying the electrodes, the subject was told to gaze toward the left and right. Thereby adjustment of the connection of electrodes was made in such a way that on the rightward gaze, E.N.G. tracings were shown as upward deflections and on the leftward gaze, they were recorded as downward deflections. In rabbits, on the rightward rotatory stimulation, the fast phase of nystagmus was recorded upward above the base line during rotation and downward below the base line after stopping rotation. By this adjustment, tracings of the rightward nystagmus appeared upward and the leftward nystagmus downward as in the case of human experiments.

In human experiments, the vision of the subject was eliminated by means of Frenzel's eyeglasses. He was told to close his eyes lightly in order to avoid the influence of the electromyogram. The subject was seated on the electric rotatory chair with the head tilted 30 degrees forward. In rabbits the animal, was enclosed and fixed in a metal cylinder with the eyes covered by a black cloth. The cylinder was placed on the electric rotatory chair and fixed in such a position that the center of a line connecting bilateral external auditory meatus of the rabbit was on the axis of rotation.

The electric rotatory chair, shown in Figure 2, was made on the following principle. Optional angular velocity may be obtained

by altering the radius of two discs which are mutually perpendicular. By turning the handle of the transmission gear, the radius of two discs can be altered, which leads to changes in the angular velocity. At a rate of one turn of the transmission gear handle in one second the chair turns at an accelerative angular velocity of $2.4^{\circ}/sec^2$. By turning the handle at constant angular velocity, the chair can be turned at a constant accelerative angular velocity. The constant angular velocity rotation of the chair can be performed automatically by pushing down the starting pedal after setting the angular velocity indicator at the desired velocity on the dial. After cessation of the stimulation E.N.G. was recorded in a dark and quiet environment, since vision and noise were known to exert a great influence upon the development of secondary nystagmus.

B. Results of Experiment

1. Incidence of Secondary Nystagmus

a. Humans

Human experiments were conducted using healthy adults free from abnormalities of the vestibular organ.

Rotatory stimulation at constant angular velocity of one turn in two seconds ($180^{\circ}/sec$) were given and the relationship between the number of rotations and the incidence of secondary nystagmus was studied. As shown in Table 1, among the 20 subjects, 3 cases (15%) developed secondary nystagmus after 5 turns or less, 9 cases (45%) after 6 to 10 turns, and 5 cases (25%) after 11 to 15 turns. Thus, in 17 out of 20 subjects (85%), secondary nystagmus appeared after 15 turns or less. In the three cases who required 16 turns or more to develop secondary nystagmus, two of them had shown equivocal tracings with an unstable baseline on the weaker stimulation. Figure 3 illustrates an example of E.N.G. in a case who developed secondary nystagmus after 15 turns (30 seconds) at a rate of one turn in two seconds.

In the next step of the experiments, the incidence of secondary nystagmus after rotatory stimulation of constant accelerative angular velocity was studied. Using various accelerative angular velocities ($2.4^{\circ}/sec^2$, $1.6^{\circ}/sec^2$, $1.2^{\circ}/sec^2$, $0.8^{\circ}/sec^2$, and $0.6^{\circ}/sec^2$) the minimal rotatory stimuli to induce secondary nystagmus were determined in terms of the terminal angular velocity by changing the duration of stimulation. The results are shown in Figure 4 where the ordinate indicates the terminal angular velocity (degree/second) and the abscissa indicates the duration of stimulation (second). Sloped lines indicate the accelerative angular velocity at $2.4^{\circ}/sec$,

1.6°/sec, 1.2°/sec, 0.8°/sec, and 0.6°/sec from left to right respectively. Open circles on the sloped lines represent the number of cases who first developed secondary nystagmus at the specified terminal angular velocity. For example, after rotatory stimulation at constant accelerative angular velocity of 2.4°/sec for 20 seconds, three cases first developed secondary nystagmus and the terminal angular velocity on the cessation of stimulation was 48°/sec. Figure 5 illustrates E.N.G. tracings of a case who underwent the stimulation at constant accelerative angular velocity of $0.6°/sec^2$ for 50 seconds. The terminal angular velocity was 30°/sec. In Table 2, the percentage incidence of secondary nystagmus is shown by terminal angular velocity and by accelerative angular velocity. At every accelerative angular velocity, secondary nystagmus was induced in 100% when the terminal angular velocity was 90°/sec. Summing up the results at each accelerative angular velocity, the incidence of secondary nystagmus were 32%, 81%, and 100% for the terminal angular velocity of 30°/sec, 60°/sec, and 90°/sec respectively. When the terminal angular velocity is equal, secondary nystagmus tends to be induced more frequently by stimulation at a smaller accelerative angular velocity. In other words, a longer duration of rotatory stimualtion gives a better chance to bring about secondary nystagmus.

b. Rabbits

Matured rabbits, particularly black or brown rabbits, were used for the experiment since it had been known that the vestibular reaction was more sensitive in black or brown rabbits than in white ones. As in human experiments, the incidence of secondary nystagmus after rotatory stimulation of both constant angular velocity and constant accelerative angular velocity was studied.

In Table 3, the incidence of secondary nystagmus after rotatory stimulation at constant angular velocity is shown. Using various combinations of different rotation velocities and durations of stimulations, secondary nystagmus was observed only in 15 out of 51 cases (28%).

In Table 4, the incidence of secondary nystagmus after rotatory stimulation at a constant accelerative angualr velocity of $1.2°/sec^2$ and $2.4°/sec^2$ are shown. When the terminal angular velocity was 60°/sec or less, secondary nystagmus was observed in no case ; between 60°/sec and 120°/sec it was induced in 3 out of 7 cases; and between 120°/sec and 180°/sec, in 3 out of 8 cases. Thus, secondary nystagmus could be induced in only 6 out of 20 cases after rotatory stimulation at the terminal angular velocity of up to 180°/sec using the stimulation at constant accelerative angualr velocity of $1.2°/sec^2$ and $2.4°/sec^2$. These results suggest that secondary

nystagmus can be induced much less easily by rotatory stimuli in rabbits than in human.

2. Vision and Incidence of Secondary Nystagmus

a. Humans

The influence of vision on the vestibular reaction was studied. First, the minimal stimulation required to induce secondary nystagmus, namely the threshold value, was determined by giving rotatory stimulation at constant accelerative angular velocity with the subjects's eyes completely covered and in a quiet room. Then after disappearance of the vestibular reaction, the same threshold stimulation was repeated. On the cessation of stimulation, the development of secondary nystagmus was examined with the subject's eyes uncovered in the light. In this experiment, none of the subjects developed secondary nystagmus under the open vision. In the experiment previously mentioned, secondary nystagmus occurred in 85% of the normal subjects under the closed vision when rotatory stimulation was given at constant angular velocity of one turn in two seconds for 30 seconds. Under the closed vision, however, the same stimulation could induce secondary nystagmus only in one out of six subjects. This result is consistent with that of Honsha's study (17). He suggested that the minimal stimulation for secondary nystagmus was a rotatory stimulation at constant angular velocity of one turn in two seconds for 60 seconds. In Table 5, examples of the effect of vision are shown. In example A, rotatory stimulation at constant accelerative angular velocity ($0.4^{\circ}/sec^2$) was given for 50 seconds. The rotation was stopped at the terminal angular velocity of 20°/sec. Under the open vision, secondary nystagmus did not occur at all whereas under the closed vision, it was overserved five times. In example B, rotatory stimulation at constant angular velocity (one turn in two seconds) was given for 60 seconds. In this case, secondary nystagmus occurred under the open vision, but its frequency and duration were much less than those under the closed vision.

b. Rabbits

As mentioned before, secondary nystagmus is much less frequently induced by rotatory stimulation in rabbits than in humans. Those rabbits which developed secondary nystagmus under the closed vision were studied. In all of them, secondary nystagmus did not occur under the open vision.

3. Repetitive Stimulation and Incidence of Secondary Nystagmus

a. Short-time Cumulative Stimulation

Repetitive rotatory stimuli, both constant accelerative angular

velocity and constant angular velocity, were given to healthy persons cumulatively in a short period of time. Rotatory stimulations of the same strength and in the same direction were repeated five times immediately after disappearance of secondary nystagmus on E.N.G. which had been induced by the previous stimulation. Table 6 shows an example in which rotatory stimulation was given at constant angular velocity of one turn in two seconds for 30 seconds. Both post-rotatory nystagmus and secondary nystagmus showed some decrease in duration with an increase in the number of stimulations. Table 7 shows an example in which rotatory stimulation at constant accelerative angular velocity with the terminal angular velocity of 28°/sec was given. No appreciable changes were observed in either the frequency or duration of secondary nystagmus.

In the whole experiment, 2 cases showed no changes and 3 cases showed a tendency of decrease in the frequency and duration of secondary nystagmus by repetitive stimulations of constant angular velocity rotation while none of the subjects showed appreciable changes by constant accelerative angular velocity stimulations.

b. Daily Stimulation

The threshold stimuli for secondary nystagmus were given to healthy persons once a day for five days. In none of the five subjects studied was there observed any consistent trend of increase or decrease in the frequency and duration of secondary nystagmus.

4. Brain Damage and Secondary Nystagmus

The basic reflex arc of the vestibular nystagmus is formed by the peripheral labyrinth--vestibular nerve fibers--vestibular nuclei--posterior longitudinal bundle--oculomotor nuclei--oculomotor nerve fibers--ocular muscles. Also, the nystagmus reaction is regulated by the complex control circuit of the cerebrum and cerebellum. The relationship of this higher control circuit to the development of secondary nystagmus was studied in the following experiments using rabbits.

a. Removal of Cerebral Cortex

When the unilateral cerebral contex was removed, the post-rotatory nystagmus showed directional preponderance to the affected side and the second phase of nystagmus also frequently occurred in the same direction. Usually, the directional preponderance was most prominent between the third and fifth post-operative days. Thereafter, the balance between left- and right-directed nystagmus was gradually restored and the directional preponderance disappeared within 10 to

20 days after the operation. The occurrence of secondary nystagmus also decreased in frequency after disappearance of the directional preponderance. Figure 6a shows the course of changes in the frequency of nystagmus after removal of the right cerebral cortex. By giving rotatory stimulation at constant angular velocity of one turn in two seconds, post-operative preponderance of the nystagmus to the right was observed. Secondary nystagmus which had not been observed before the operation occurred in the same direction from the second post-operative day.

b. Removal of Cerebral Hemisphere

When the unilateral cerebral hemisphere was removed, marked directional preponderance to the affected side was observed. Spontaneous nystagmus toward the affected side continued after the operation for about seven days in mild cases and for almost one month in severe cases. After cessation of the continuous spontaneous nystagmus, spontaneous nystagmus could be provoked easily by rotatory stimulation of one turn in two seconds for 30 seconds.

When the bilateral cerebral hemisphere was removed, the attack was so severe that the rabbits rarely survived. Figure 6b shows an example of this procedure. In this case, the frequency of nystagmus during and after rotatory stimulation markedly decreased. On the other hand, secondary nystagmus appeared more frequently.

c. Removal of Temporal Region

Anatomical classification of the cerebral cortical areas in rabbits is controvertial. In this experiment, the areas 20, 21, and 22 according to the Winkler-Potter classification were removed. Like the case of decortication, directional preponderance to the affected side was observed and secondary nystagmus was apt to occur in the same direction.

d. Removal of Parietal Region

As the frontal region of rabbits is poorly developed, it was often damaged when the frontal region was removed. In most cases, therefore, the areas 4.1 + 3.5 + 7 were completely damaged. In such a case, mild directional preponderance to the affected side and secondary nystagmus in the same direction were observed.

e. Removal of Cerebellar Paraflocculer Lobe

When unilateral paraflocculer lobe of the cerebellum was removed, marked directional preponderance to the affected side was

observed in every case. This directional preponderance became most prominent on the fourth and fifth days after the operation. Thereafter it gradually decreased in severity and the balance between the left- and right-directed nystagmus was restored 10 to 15 days after the operation. Figure 6c shows an example showing the peak of directional preponderance on the fifth day after removal of the paraflocculer lobe.

In summary, when the directional preponderance is induced by experimental brain damage, secondary nystagmus is likely to occur in the same direction as that of directional preponderance.

C. Summary and Discussion of Part I

The minimal stimulus to induce the secondary phase of post-rotatory nystagmus has been studied by many investigators. There is no agreement, however, on the threshold value of secondary nystagmus. The results obtained in this study are compared with other reports as follows:

Lange (1939)[10] stated that the terminal angular velocity required to induce secondary nystagmus varied between 30°/sec and 120°/sec when rotatory stimulation was given at constant accelerative angular velocity of 0.5°/sec^2 to 1.0°/sec^2 . He also pointed out that the threshold value of secondary nystagmus decreased on the side of "Nystagmusbereitschaft" [Nystagmus preparedness] when the central tonus difference was present. Based on this finding, he postulated the involvement of central factors in the development of secondary nystagmus. Mittermaier (1955)[11] reported that induction of secondary nystagmus required stronger stimuli than those needed to induce post-rotatory nystagmus (first phase) and that the threshold value of secondary nystagmus was difficult to determine since the vestibular nystagmus itself was variable under the same condition. In his study, the incidence of secondary nystagmus was quite variable depending on accelerative angular velocity: 65% for 1.3°/sec^2, 75% for 1.6°/sec^2, 85% for 2.3°/sec^2, and 90% for 2.6°/sec^2 when the terminal angular velocity was 60°/sec; and 100% for 3.0°/sec^2 when the terminal angular velocity was 60°/sec. He thus postulated that the occurrence of secondary nystagmus was related to the duration of rotatory stimulation but not to the physical energy of rotation. Schmalz (1932)[13] stated the same opinion based on the fact that rotatory acceleration is a relative acceleration related to acceleration of the semicircular canal and to the density and viscosity of endolymph within the semicircular canal. Aschan and Bergstadt (1955)[2] reported that secondary nystagmus occurred in one-third of the tested subjects when rotatory stimulation at the accelerative angular velocity

of $3.0^{\circ}/sec^2$ was for 10 seconds; in no case at $4.0^{\circ}/sec^2$ for 6 seconds; and in all cases at $2.0^{\circ}/sec^2$ for 25 seconds. They also stated that the occurrence of secondary nystagmus was unrelated to the theoretically calculated deviation of the Cupuda but related to the duration of the first phase of post-rotatory nystagmus. They concluded that secondary nystagmus was more apt to be induced by weak rotatory stimuli such as $1.0^{\circ}/sec^2$ given for a long period of time. In Japan, there is no literature which studied the threshold value of secondary nystagmus. By means of E.N.G. Honsho (1934)[17] reported the results of visual observations of nystagmus using manual rotatory chair. He found that secondary nystagmus occurred in 17% after 45 turns of the chair at standard rate (one turn in two seconds), in 50% after 60 turns, and in 80% after 75 turns.

Comparing the results obtained in this study with those in Mittermaier's study, the threshold value is lower than his value. Namely, in the present study the terminal angular velocity of $90^{\circ}/sec$ was required regardless of accelerative angular velocity to induce secondary nystagmus in 100% of the subjects whereas in Mittermaier's study it was $60^{\circ}/sec$ at accelerative angular velocity of $3.0^{\circ}/sec^2$. The difference seems to be due to different methods of recording in the two studies.

It has been well known that the vision exerts a great influence on the vestibular nystagmus. The same applies secondary nystagmus. Aschan (1955) reported that the duration of post-rotatory nystagmus under the open vision was 85% shorter than that under the closed vision using Frenzel's eyeglasses. Mahony (1957) stated that spontaneous nystagmus after rotatory stimulation was often prevented from occurring or disappeared by opening the eyes, but reappeared by closing the eyes. However, Ohm (1939), Aschan (1955)[3], Aschan and Bergstadt (1955)[2], and Henriksson (1955) [9] insist that elimination of vision by using Frenzel's eyeglasses may have a considerable influence on the vestibular nystagmus but is insufficient to eliminate fixation. In the present study, observations of nystagmus were performed in a dark and quiet room to eliminate the effect of vision and noise. Nevertheless, the threshold stimuli could not induce secondary nystagmus under the open vision. When Frenzel's eyeglasses were used, secondary nystagmus was recognized only occasionally. These findings obviously indicate the great influence of vision on the development of secondary nystagmus. It is important, therefore, to eliminate the vision in studying secondary nystagmus.

Concerning the factors of stimulation to induce secondary nystagmus, Fisher postulated the accelerative angular velocity and terminal angular velocity, Honsho claimed the amount of stimulation,

and Aschan insisted the duration of the first phase of post-rotatory nystagmus. The author favors Fisher's opinion and assumes that the terminal angular velocity has the greatest effect on the development of secondary nystagmus regardless of accelerative angular velocity.

The threshold value of secondary nystagmus is not constant even in the same individual. This might be related to physical and emotional factors of the subject in addition to the conditions of recording and stimulation apparatus.

Based on the brain damage experiments in rabbits, it is concievable that secondary nystagmus is brought about by the basic reflex arc of vestibulal nystagmus and is also controlled by the higher regulatory circuit of the cerebrum and cerebellum.

D. Conclusions of Part I

1. In healthy persons, when rotatory stimulation at a constant angular acceleration of $0.6^{\circ}/sec^2$ to $2.4^{\circ}/sec^2$ was given until the terminal angular velocity reached $90^{\circ}/sec$, secondary nystagmus was induced in all of the subjects tested. The occurrence of secondary nystagmus seems to be related to the terminal angular velocity. The minimal value for the terminal angular velocity to induce secondary nystagmus, however, was not constant in the same individuals.

2. When rotatory stimulation at a constant angular velocity of one turn in two seconds was given, 85% of the subjects developed secondary nystagmus after 15 turns or less.

3. In rabbits, the incidence of secondary nystagmus was much lower than in humans, being 30% on the average.

4. In the presence of free vision, the incidence of secondary nystagmus decreased remarkably.

5. Repetition of the stimulation did not appreciably alter the incidence of secondary nystagmus.

6. In the presence of unbalanced nystagmus due to brain damage, secondary nystagmus was more apt to occur in the same direction as that of directional preponderance.

Part I

Figure 1 Electronystagmographic apparatus

Figure 2 Electric rotatory chair

a. [Illegible, possibly original wave]

b. [Illegible, possibly differential wave]

Figure 3 Electronystagmogram of rotatory secondary nystagmus in a healthy person (clockwise rotation at constant angular velocity of 1 turn in 2 seconds for 30 seconds)

a. Terminal angular velocity

b. Duration of stimulation

Figure 4 Distribution of incidence of rotatory secondary nystagmus in healthy persons (rotation at constant accelerative angular velocity)

a. [Illegible; possibly, onset]

b. [Illegible; possibly, cessation]

c. [Illegible; possibly, differential wave]

d. [Illegible; possibly, original wave]

e. [Illegible; possibly, secondary phase]

Figure 5 Electronystagmogram of rotatory secondary nystagmus in a healthy person

a. Removal of right cerebral contex
____ Nystagmus to the right
.....Nystagmus to the left

b. Removal of bilateral cerebral hemispheres
_____ N. to the right
..... N. to the left

c. Removal of right cerebella paraflocculus lobe
____ N. to the right
.... N. to the left

d. Frequency of N. during rotation

e. Frequency of post-rotatory N.

f. Frequency of secondary N.

Figure 6

Table 1. Incidence of rotatory secondary nystagmus in healthy persons (constant angular velocity of 1 turn in 2 seconds)

Number of rotation	5 or less	6-10	11-15	16 or more	Total
Number of subjects developing secondary nystagmus	3	9	5	3	20 cases
Percentage	15	45	25	15	100

Table 2. Incidence of rotatory secondary nystagmus in healthys persons (constant accelerative angular velocity)

a. Terminal angular velocity b. Acceleration c. Total

Table 3. Incidence of rotatory secondary nystagmus in rabbits (constant angular velocity)

a. duration b. velocity c. total d. 60 sec or less e. seconds

Figures indicate number of rabbits tested and figures in parenthesis indicate number of cases developing secondary nystagmus

Table 4. Incidence of rotatory secondary nystagmus in rabbits (constant accelerative angular velocity)

a. terminal angular velocity b. acceleration c. 60°/sec or less
d. total

Figures indicate number of rabbits tested and figures in parenthesis indicate number of cases developing secondary nystagmus

Table 5. Rotatory secondary nystagmus and vision in healthy persons

(A) Threshold stimulation

(B) Rotation at constant angular velocity

a. stimulation b. vision c. post-rotatory nystagmus d. frequency
e. duration f. resting period g. secondary nystagmus h. closed
i. open j. 30 years male k. 24 years female

Table 6. Cumulative stimulation and rotatory secondary nystagmus in healthy persons (rotation at constant angular velocity of 1 turn for 30 seconds)

a. number of stimulation b. post-rotatory nystagmus c. frequency
d. duration e. resting period f. secondary nystagmus

Table 7. Cumulative stimulation and rotatory secondary nystagmus in healthy persons (rotation at constant accelerative angular velocity of $0.4^{\circ}/sec^{2}$ and terminal angular velocity of $28^{\circ}/sec$)

PART II: THE SECOND PHASE OF CALORIC NYSTAGMUS

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Introduction

Unlike the second phase of post-rotatory nystagmus (rotatory secondary nystagmus), that of caloric nystagmus (caloric secondary nystagmus) has not been studied extensively. There are only fragmentary reports on this phenomenon by Thronval (1936), Takahashi (1952), Jung (1953), Aschan and Bergstadt (1955), Stahl (1950), Hamerama (1957), McLay and Madigan (1957), and Wagemann (1958). Furthermore, almost all of the reported caloric secondary nystagmus are induced only by simultaneous stimulation of cold and hot water. For this reason Aschan explained the occurrence of caloric secondary nystagmus by a central process in response to cumulative stimuli, and Jung attributed it to a mechanism of the post-excitatory suppression phase. On the other hand, Takahashi, Wagemann and McLay and Madigan observed caloric secondary nystagmus induced by unilateral cold water stimulation. These observations were made, however, using a method of long-time irrigation, which may not be generally applied to caloric stimulation.

The present study was attempted to induce secondary nystagmus with a high probability on the ordinary caloric stimulation by utilizing improved techniques of observation and stimulation and to elucidate the causal mechanism of this phenomenon.

Since caloric nystagmus, like post-rotatory nystagmus, is a reaction of the vestibulo-ocular reflex, it could not be expected to observe the secondary phase if the role of vision is ignored. Also, the maintenance of constant temperature of the water to be used for stimulation and the duration of irrigation are important factors as reported by Jongkees, Aschan, etc. In the present study these factors were taken into serious consideration to carry out the experiments.

A. Methods of Experiment

On the performance of caloric stimulation, particular attention was paid to such factors as constant temperature of water, amount of water, duration of irrigation, and position of the head. The amount of water (20 cc), duration of irrigation (7-10 seconds), and position of the head (30 degrees forward tilt) were kept constant and the degree of stimulation was adjusted only by changing the temperature of water. The reason for determining the amount of water as 20 cc is to lessen the burden of test subjects and to keep the influence of changes in the temperature of water during irrigation minimal. Within the amount of 20 cc, changes in the amount of thermic energy

resulting from altered temperature by 1° C would be neglibible and hence the influence of temperature changes of 1-2° C could be ignored.

Caloric stimualtion was performed by irrigating the ear with cold water at a given temperature from an electric constant temperature water circulating bath. The temperature of water was checked before entering the ear for the difference from the temperature within the bath. The reason for using cold water is that cold water produces a stronger stimulation than hot water when the differences between body temperature and both cold and hot water are equal, according to Aschan (1955) and Bárány (1907).

In order to eliminate the influence of vision, noise, and excitement, observations of nystagmus after caloric stimulation were carried out in a dark and quiet room as in the experiment of rotatory secondary nystagmus.

Methods of application of electrodes, elimination of vision and recording of E.N.G. were the same as described in Part I. The test rabbits were enclosed and fixed in a cylinder of which the longitudinal axis made an angle of 30 degree against horizontal plane.

B. Results of Experiment

1. Incidence of Caloric Secondary Nystagmus

a. Humans

Healthy adult males and females having normal auditory organs and vestibular fractions were used for the experiment. The maximal temperature of water (20 cc) to induce caloric secondary nystagmus was determined by using different temperature between 35°C and 15° C. The threshold value of caloric secondary nystagmus was tentatively represented by the difference between body temperature of the subject and the maximal temperature of water to induce caloric secondary nystagmus. As shown in Table 1, caloric secondary nystagmus was induced in 3 out of 22 cases (13%) by cold water stimulation when the difference between body temperature and water temperature was 5° C or less, in 4 cases (18%) when the difference was between 5° C and 10° C, in 6 cases (27%) when the difference was between 10°C and 15° C, and in 3 cases (11%) when the difference was between 15° C and 20° C. In other words, caloric secondary nystagmus was elicited by unilateral caloric stimulation using 20 cc of cold water of which temperature differed from body temperature by 20° C or less in 16 out of 22 subjects (72%). Those 6 cases in which secondary nystagmus was not clearly observed were found to have some technical reasons

for this failure. It is expected, therefore, to be able to induce caloric secondary nystagmus in 100% when recording apparatus and techniques are more improved. Figure 1 illustrates an example of E.N.G. recorded for a 24-year old woman whose left ear was irrigated with 26° C water. Her body temperature was 35.6° C. After a latent period of about 6 seconds following the onset of irrigation, caloric nystagmus developed. It lasted for 163 seconds showing upward deflections on the tracing. Then, following a resting period of 21 seconds, secondary nystagmus occurred showing deflections in the opposite direction (downward) and continued for 133 seconds.

b. Rabbits

Black or brown rabbits were used for the same reason described in Part I. Caloric stimulation was carried out using cold water of which temperature differed from body temperature by 30° C or less. The incidence of caloric secondary nystagmus is shown in Table 2. Secondary nystagmus was induced in 3 out of 22 cases (13%) when the temperature difference was 15° C or less, in 5 cases (24%) when the difference was between 15° C and 25° C, and in 9 cases (40%) when the difference was between 25° C and 30° C. Thus, in rabbits, caloric secondary nystagmus was elicited by unilateral caloric stimulation using 20 cc of cold water of which temperature differed from body temperature by 30° C or less in 17 out of 22 rabbits (78%).

2. Course of Caloric Secondary Nystagmus

The degree of caloric nystagmus reaction to the unilateral irrigation with cold water varies depending on the individual's constitution and the strength of stimulation. Table 3 shows a part of the results obtained from healthy persons on whom the threshold stimulation for caloric secondary nystagmus was performed. The average threshold value of 16 subjects was 12° C. The average latent period (interval between the developemnt of the first phase of caloric nystagmus and the onset of irrigation) was 13 seconds. The average frequency of this nystagmus was 125 and the average duration was 175 seconds. In many cases, however, the end point of the first phase could not be determined clearly since both frequency and speed of this nystagmus gradually tapered off. Therefore, a transient period showing sporadic appearance of both first and second phases of nystagmus was tentatively called "resting period" and the point at which time deflection in the opposite direction to the first phase became clearly increasing was assumed to be the onset of secondary nystagmus. The amplitude of secondary nystagmus was generally smaller than that of the first phase, and so was the frequency. Toward the end of secondary nystagmus the amplitude of deflection became very small and

the interval between each deflection became longer, and finally it disappeared completely.

It was impossible to predict the occurrence of secondary nystagmus from the frequency, duration, or latent period of the first phase of caloric nystagmus. Also, there was no uniform relationship between the strength of coloric stimulation and the frequency or duration of both first and second phases of nystagmus.

Table 4 shows the results of an experiment using different temperatures of water for stimulation in the same individual. There was a definite tendency of increase in both frequency and duration of secondary nystagmus with decrease in the temperature of water (increase in the difference between body temperature and temperature of cold water). In this particular case, however, the stimulation was repeated immediately following disappearance of the previous reaction. Therefore, the effect of cumulative stimuli can not be ruled out.

3. Relationship Between Caloric Secondary Nystagmus and Post-Rotatory Secondary Nystagmus

a. Threshold Values

Comparing rotatory stimulation and caloric stimulation to the vestibular organ, one of the basic differences is that the former is bilateral stimulation, whereas the latter is an unilateral one. However, the effect of both stimulation on the vestibular function would not completely be unrelated. On the basis of this assumption, threshold values of rotatory and caloric secondary nystagmus were compared in ten healthy persons. First, the threshold value of rotatory secondary nystagmus was determined in terms of the terminal angular velocity using rotatory stimualtion at a constant accelerative angular velocity of $0.4^{o}/sec^{2}$. At an interval of 30 minutes following the completion of rotatory test, the caloric test was performed by irrigating the ear with 20 cc of cold water for 10 seconds to determine the threshold in terms of the difference between body temperature and temperature of the water. The results are illustrated in Figure 2. The ordinate indicates the threshold value of rotatory secondary nystagmus (^{o}/sec) and the abscissa indicates that of caloric secondary nystagmus (^{o} C). Open circles connected by solid lines represent threshold values of both rotatory and caloric secondary nystagmus in individual subjects. The existence of a close correlation between the two threshold values is clearly shown in this figure. The correlation coefficient (r) was computed by the following equation, where Xi is the threshold value of caloric secondary nystagmus and Y_i is that of rotatory secondary nystagmus.

Xi .... 3, 5, 6, 8, 10, 13, 14, 18, 19, 20° C

Yi .... 18, 22, 20, 20, 40, 48, 44, 54, 44, 64 (°/sec)

$$r = \frac{\frac{1}{N}\sum^{N}(Xi - \bar{X})(Yi - \bar{Y})}{\delta_x \cdot \delta_y}$$

$$= \frac{77.28}{6.11 \times 15.78} = 0.8095 \fallingdotseq 0.81$$

Thus, the correlation coefficient between the two threshold values was 0.81 indicating a close correlation.

As described in Part I, the threshold value of rotatory secondary nystagmus varies day by day in the same individual. Whether or not this applies the threshold value of caloric secondary nystagmus was studied by performing three consecutive caloric stimulations at an interval of more than 24 hours for the same individual. The results are shown in Table 5. The threshold value of caloric secondary nystagmus as well as of rotatory secondary nystagmus varied on different days. Besides technical factors such as recording instrument and measurement errors, some personal factors might be responsible for this variablity. The answer to this question must await future studies.

b. Relationship between Frequency of Post-Rotatory Nystagmus and Threshold Values of Rotatory and Caloric Secondary Nystagmus

Using the same subjects of the foregoing experiment, the relationship between the frequency of post-rotatory nystagmus (first phase) and threshold values of rotatory and caloric secondary nystagmus was studied. As represented in Figure 2 by cross marks connected by dotted lines, the frequency of post-rotatory nystagmus showed a trend opposite to that of threshold values. In other words, subjects showing a tendency of hypernystagmus (greater frequency of post-rotatory nystagmus) had lower threshold values of secondary nystagmus.

c. Relationship between Long-Time Irrigation and Rotatory Nyatagmus

Rotatory nystagmus was studied in rabbits before and after

unilateral long-time irrigation of the ear with cold water. A plastic tube of 1 mm in diameter was inserted into the external auditory canal, which was connected with a rubber tube from an electric constant temperature water circulating bath. The tip of the plastic tube was placed about 3 mm apart from the tympanic membrane. The irrigation was performed with cold water at 15° C and at a rate of 10 cc per 10 seconds. The stimulation was given for varying lengths of duration; 15 minutes, 45 minutes, and 100 minutes. In Table 6 are the results of an experiment using the right ear stimulation with 15° C water for 15 minutes. During irrigation no changes in the direction and frequency of nystagmus were observed. Following disappearance of E.N.G. evidence of the vestibular reaction to irrigation, rotatory stimulation was given and rotatory nystagmus was compared with that before irrigation. During the rotatory stimulation (left turn) after irrigation, nystagmus to the left first occurred 31 times in 13 seconds. Then, following a period of pause for 11 seconds, nystagmus to the right occurred 7 times in 6 seconds until cessation of the stimulation. Table 7 shows results of an experiment using the left ear stimulation with 15° C water for 45 minutes. During irrigation, no changes in the direction of nystagmus were observed and after irrigation, the same nystagmus continued for two and one half hours. Post-rotatory nystagmus after irritation shows a slight preponderance of the right-directed nystagmus, and also secondary nystagmus which had not been observed before irrigation was elicited. Table 8 shows results of an experiment using the right ear irrigation with 15° C water for 100 minutes. During irrigation, no changes in changes in the direction of nystagmus occurred but periodic changes in the frequency and amplitude of nystagmus of a brief period (1-3 minutes) were observed after 70 minutes following the onset of irrigation. The right-directed rotatory nystagmus was more apt to occur after irrigation and secondary nystagmus was first observed after irrigation.

d. Effect of Brain Damage on Rotatory and Caloric Secondary Nystagmus

The following experiments were carried out to see the effect of brain damage causing liability of rotatory secondary nystagmus on the occurrence of caloric secondary nystagmus.

(i) Removal of Unilateral Cerebral Cortex

Unilateral cerebral decortication was performed using an electric suction apparatus. Table 9 shows results on the third day following the operation. Both rotatory and caloric secondary nystagmus were more apt to occur in the direction of "directional preponderance" (the operated side).

(ii) Removal of Unilateral Cerebellar Paraflocculus Lobe

Table 10 shows results of the removal of unilateral cerebellar paraflocculus lobe. As in the cerebral decortication, both rotatory and caloric secondary nystagmus were induced more readily in the direction of the operated side.

4. Effect of Repetitive Stimulation

When homolateral caloric stimulation with cold water was given consecutively in rabbits, secondary nystagmus tended to occur more readily with an increase in the number of stimulations. The duration of the resting period did not show a tendency of either prolongation or shortening. Table 11 shows an example in which five consecutive stimulation were given using 400 cc of cold water at 15° C to the right ear.

In humans there was a tendency of shortening of the resting period as shown in Table 12.

C. Summary and Discussion of Part 2

The main reasons for relatively infrequent observations of the secondary phase of caloric nystagmus in the past are considered to be improper and inadequate methods of recording and observation. The present study was carried out with due care using an electronystagmographic machine provided with differential circuit under the condition of complete elimination of vision and noise. By this method, caloric secondary nystagmus could be induced in almost all cases of healthy persons except a few subjects whose E.N.G. was difficult to read because of an unstable base line. Takahashi (1952), Wagemann (1958), and McLay and Madigan (1957) reported the observation of caloric secondary nystagmus induced by unilateral cold stimulation. However, these observers had to use long-time irrigation to elicit secondary nystagmus. Takahashi attributed the occurrence of secondary nystagmus to paralysis of the vestibular organ. Aschan and Bergstadt (1955) reported that caloric secondary nystagmus was induced only by simultaneous stimulation of one ear with cold water and the other ear with hot water. They postulated a theory of central accumulation of stimuli to explain the mechanism of secondary nystagmus. On the other hand, Jung (1953) described that caloric secondary nystagmus was induced by unilateral cold stimulation only in subjects with hyperexcitivity such as multiple sclerosis and attributed this phenomenon to a post-excitatory suppression mechanism. In the present study, caloric secondary nystagmus was observed in 72% of healthy persons and in 78% of rabbits.

In order to explain the phenomenon of caloric nystagmus on

the premise of Bárany's hypothesis, viscosity of the endolymph, particularly in relation to temperature, must be studied. However, very little has been done on this problem. According to Kohlraush (1947) and Gernardt (1950) changes in temperature by 1° C results in alterations of viscosity by 2.5%, and viscosity of the endolymph is two to three times that of water. In the present study, it was experienced that a 1° C difference of irrigation temperature was a definitive factor to induce caloric secondary nystagmus although no such experience was encountered for a 1° difference of the terminal angular velocity in the experiment of rotatory secondary nystagmus. This is probably due to the fact that rotatory stimulation is a simultaneous bilateral stimulation whereas caloric stimulation is a selective unilateral stimulation. In the long-time irrigation experiment, rotatory secondary nystagmus was more apt to occur after the irrigation. This might be explained by a temporary increase in the excitability of the vestibular organ.

It has been well known that the vestibular nystagmus shows directional preponderance to the operated side after removal of the unilateral cerebral cortex or cereballar paraflocculus lobe. Secondary nystagmus also tends to occur in the same direction. Hallpike (1942) assumed the presence of a "tonic controlling center" in the temporal lobe and postulated that a tendency of nystagmus toward the affected side occurred due to unbalanced control of the basic reflex arc of vestibular nystagmus resulting from pathologic lesions in the temporal lobe. Morimoto (1955)[19] and Sakai (1956)[21] attached importance to the cerebellar paraflocculus lobe in the controlling mechanism of vestibular nystagmus. The fact that both rotatory and caloric secondary nystagmus are apt to occur in the direction of directional preponderance suggests with high possibility the presence of regulatory mechanism of secondary nystagmus through the higher control circuit of the cerebrum and cerebellum.

On the other hand, from the findings of the present study showing a close correlation between threshold values of rotatory and clarolic secondary nystagmus and lower threshold values in cases with hypernystagmus, it is concievable that the peripheral labyrinth has at least secondary significance for the development of secondary nystagmus.

D. Conclusion of Part II

1. In healthy persons, caloric secondary nystagmus was induced in 72% of the subjects by unilateral stimualtion using cold water of which temperature differed from body temperature by 20° C or less. The threshold value of caloric secondary nystagmus was not constant

in the same individuals.

2. In rabbits, caloric secondary nystagmus was induced in 78% by unilateral stimulation using cold water of which temperature differed from body temperature by 30° C or less.

3. No constant relationship was found between the development of caloric secondary nystagmus and the frequency and duration of the first phase of nystagmus or the resting period.

4. There was a close correlation between threshold values of rotatory and caloric secondary nystagmus.

5. The threshold values of rotatory and caloric secondary nystagmus were low in the cases with hypernystagmus.

6. Caloric secondary nystagmus was apt to occur in the direction of directional preponderance.

E. Summary and Discussion of Whole Study

As mentioned above, secondary nystagmus can be induced by either rotatory or caloric stimulation in humans and in rabbits. This phenomenon has been over looked mainly due to inadequate methods of recording and observation. There are some difference in the incidence rate of secondary nystagmus between humans and rabbits and also between various methods of stimulation. It is expected to induce secondary nystagmus in 100% when the recording apparatus and methods are further improved. It should be noted, however, that more than a certain amount of stimulation is required to induce secondary nystagmus with a high probability and the threshold stimulation is inadequate for this purpose.

Anyway, it appears evident that secondary nystagmus should be regarded as a common feature of the vestibulal nystagmus reaction. supportive evidence have been obtained by my co-workers, Koike and Mizukoshi, in their study of optokinetic nystagmus. They performed optokinetic stimulation using a drum rotated at an acceleration of $0.5°/sec^2$ - $1.2°/sec^2$, after stopping the rotation of a drum at the terminal rugular velocity of 90°/sec or 120°/sec, E.N.G. was recorded with complete elimination of vision and in a quiet room. By this method, the incidence of optokinetic secondary nystagmus in healthy persons was 80-90%. Table 13 shows the course of optokinetic nystagmus observed in three healthy subjects. Figure 3 illustrates an example of optokinetic secondary nystagmus showing different courses of development. In one type, the first phase of nystagmus continued

for a fairly long period following cessation of the stimulation, and then after a period of pause, secondary nystagmus developed. In the other type, the first phase was not observed at all or did appear only for a few seconds, and then after a resting period, secondary nystagmus occurred. Most of the optokinetic nystagmus in healthy subjects belonged to the first type. Variability of the frequency and duration of the first phase, resting period, and of the frequency and duration of secondary nystagmus was much greater than those of the vestibular nystagmus. Inconsistency of these factors was also observed in the same individuals under the same conditions. Therefore, the effect of personal factors, physical and emotional, on the development of optokinetic secondary nystagmus cannot be ruled out.

In rabbits, the incidence of optokinetic secondary nystagmus was 100%. Table 14 shows the course of optokinetic nystagmus in rabbits. Variability of the first phase, resting period and secondary nystagmuswas also present in rabbits. Consecutive cumulative stimulation brought about tendencies of shortening of the resting period and of an increase in the frequency and duration of secondary nystagmus to some extent.

Very few reports have been made on the observation of optokinetic secondary nystagmus. Aschan (1955) described that optokinetic secondary nystagmus was observed only after optokinetic stimulation of long-time acceleration. Mizukoshi[22] reported that optokinetic secondary nystagmus was apt to occur in the direction of directional preponderance after cerebral decortication as with the vestibular nystagmus.

As to the etiologic mechanism of secondary nystagmus, there have been two antagonistic hypotheses; central origin theory by Fisher, Dodge, Schamlz, Veits, Mittermaier, Aschan, etc., versus peripheral origin theory by Groen, Jongkees, etc.

Aschan criticized Jongkee's hypothesis that the degree of deviation of the Cupula was the important causative factor of secondary nystagmus. He claimed that secondary nystagmus was caused by the central accumulation of stimuli instead of deviation of the Cupula on the basis of the following findings: (1) caloric secondary nystagmus is induced not by unilateral stimulation with cold water or bilateral simulataneous stimulation with water of the same temperature but only by bilateral simultaneous stimulation with cold and hot water; (2) unilateral cold stimulation alone is sufficient to cause deviation of the Cupula; and (3) secondary nystagmus can be induced by optokinetic stimulation which is unrelated to deviation of the Cupula. I do not think that deviation of the Cupula is essential to the development

of secondary nystagmus since secondary nystagmus was indeed in 80-90% of healthy persons and in 100% of rabbits by optokinetic stimulation in the present study.

There also is a room to doubt the validy of Jung's post-excitatory suppression theory. He attributed the occurrence of secondary nystagmus to the difference in the exitability of the left and right labyrinths resulting in a time lag of the central excitation in response to stimulation. However, optokinetic secondary nystagmus cannot be explained by this theory.

The peripheral origin theory by Groen, Jongkees, etc. follows the school of Mach-Brauer-Brown. They attempted to explain the occurrence of secondary nystagmus by some electric phenomena or chemical changes induced within the Cupula, or by deviation or deformation of the Cupula in response to the stimulation of the peripheral labyrinth. Since no details have been known concerning physical or chemical properties of the Cupula, it would be difficult to ignore completely the possibility of this hypothesis. However, optokinetic secondary nystagmus again can not explained by this mechanism as Aschan pointed out.

From the foregoing discussion of various hypotheses on the origin of secondary nystagmus, it seems evident that the causal mechanism of secondary nystagmus is not simple enough to be explained by a single factor. My assumption based on the results obtained from the present study is as follows. Secondary nystagmus is not a phenomenon unique to the vestibular reaction after rotatory or caloric stimulation but is a basic pattern of nystagmus reaction common to all rotatory, caloric and optokinetic nystagmus. Failure of the observation of secondary nystagmus is considered a result of interference by vision and other suppressive factors. Taking account of the fact that the occurrence of secondary nystagmus varies in the same individuals under the same conditions and that cerebral or cerebellar decortication can alter the incidence of secondary nystagmus, it is assumed that the physiologic mechanism of secondary nystagmus belongs to the basic reflex arc of nystagmus under the regulation of the higher control pathways including the temperal lobe and cerebellum.

F. Conclusions of Whole Study

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Experiments were carried out on the secondary phase of nystagmus using healthy persons and rabbits. The following conclusions were obtained.

1. The secondary phase of nystagmus is a basic pattern of the nystagmus

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reaction common to rotatory, caloric, and optokinetic nystagmus.

2. The second phase of nystagmus is a function of the basic reflex arc of nystagmus reaction and is regulated by the higher control pathways involving the cerebrum and cerebellum.

AUTHOR

a. [Illegible; possibly, original wave]

b. [Illegible; possibly, differential wave]

Figure 1. Course of caloric nystagmus in a healthy person (left ear, 26°C, 20 cc)

a. Threshold value of rotatory secondary nystagmus (terminal angualr velocity)
b. Threshold value of caloric secondary nystagmus (difference between body temperature and water temperature
c. o— threshold value of secondary nystagmus
d. x···· frequency of post-rotatory nystagmus at acceleration of $0.4°/sec^2$ and terminal angular velocity of $20°/sec$

Figure 2. Threshold values of rotatory and caloric secondary nystagmus and frequency of post-rotatory nystagmus

a. [Illegible]

b. [Illegible]

c. [Illegible; possibly, secondary phase]

Figure 3. Electronystagmogram of optokinetic nystagmus in a healthy person

Table 1. Incidence of caloric secondary nystagmus in healthy persons

a. difference between body temperature and water temperature b. number of subjects developing secondary nystagmus c. percentage (out of 22 cases) d. 5° or less e. total f. cases

Table 2. Incidence of caloric secondary nystagmus in rabbits

[Same legend as in Table 1.]

a. subjects b. temperature difference c. caloric nystagmus d. latent period e. frequency f. duration g. resting period h. secondary nystagmus i. average of 16 cases

Table 4. Caloric nystagmus by different amount of stimulation in the same individual (healthy person)

a. order of stimulation b. temperature difference c. stimulated ear
d. caloric nystagmus e. latent period f. frequency g. duration
h. secondary nystagmus i. resting period j. right

Table 5. Variability of secondary nystagmus in the same individual (healthy person)

a. threshold value b. caloric secondary nystagmus (difference between body temperature and water temperature ^{o}C) c. rotatory secondary nystagmus (terminal angular velocity $^{o}/sec$) d. date of experiment

Table 6. Rotary nystagmus in a rabbit before and after irrigation of the right ear with cold water at 15° C for 15 minutes (rotation at constant angular velocity of 1 turn in 2 seconds for 30 seconds)

a. direction of rotation b. before or after irrigation c. frequency of nystagmus during rotation d. post-rotatory nystagmus e. frequency f. duration g. resting period h. secondary nystagmus i. right j. left k. before irrigation l. after irrigation

Table 7. Rotatory nystagmus in a rabbit before and after irrigation of the left ear with cold water at 15° C for 45 minutes (at 1 turn in 2 seconds, for 30 seconds)

[Same legend as in Table 6.]

Table 8. Rotatory nystagmus in a rabbit before and after irrigation of the right ear with cold water at 15° C for 100 minutes (at 1 turn in 2 seconds, for 30 seconds)

[Same legend as in Table 6.]

Table 9. Nystagmus before and after removal of the right cerebral cortex in rabbits

(A) Rotatory nystagmus (stimulation: 1 turn in 2 seconds, for 30 seconds)

[See legend on next page.]

(B) Caloric nystagmus (stimulation: 20° C, 20 cc)

a. direction of rotation b. before or after operation c. frequency of nystagmus during rotation d. post-rotatory nystagmus e. resting period f. secondary nystagmus h. duration i. right j. left k. before operation l. after operation m. irrigated ear n. right ear o. left ear p. latent period q. caloric nystagmus

Table 10. Nystagmus before and after removal of the right cerebellar paraflocculus lobe

(A) Rotatory nystagmus (stimulation: 1 turn in 2 seconds, for 30 seconds)

[same legend as in Table 9.]

(B) Caloric nystagmus (stimulation: 20° C, 20 cc)

[same legend as in Table 9.]

Table 11. Effect of repetitive stimulation on caloric nystagmus (rabbit)

(Stimulation: right ear, 15° C, 40 cc)

a. order of stimulation b. caloric nystagmus c. latent period
d. frequency e. duration f. resting period g. secondary nystagmus

Table 12. Effect of repetitive stimulation on caloric nystagmus

(Stimulation: right ear, 20° C, 20 cc)

[same legend as in Table 11.]

Table 13. Course of optokinetic nystagmus in health persons

a. subjects b. stimulation c. direction of drum rotation d. post-nystagmus e. resting period f. secondary nystagmus g. frequency h. duration i. acceleration $1^{\circ}/sec^{2}$ j. terminal angular velocity $120^{\circ}/sec$ k. year-old l. right m. left

Table 14. Course of optokinetic nystagmus in rabbits

a. rabbit No. b. stimulation c. direction of drum rotation d. post-nystagmus e. resting period f. secondary nystagmus g. frequency h. duration i. acceleration $1^{\circ}/sec^{2}$ j. terminal angular velocity $120^{\circ}/sec$

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